EDITORIAL ARTICLES.

LANDERER'S MECHANICAL THEORY OF INFLAMMATION.

Inflammation has been regarded as arising from very different causes and conditions at different periods in the development of medical knowledge, some schools attributing the phenomena to changes in the blood itself, while others considered the walls of the capillaries; others, again, the nerves and muscles of the blood-vessels, to be primarily affected; and a fourth school believes inflammation due to changes occurring in the tissues surrounding the capillaries.

Thus Böerhave and Hunter held that the elements of the blood were rendered more adhesive and thus produced the initial stasis, the main feature in inflammation, just as Magendie and Poiseuille spoke of greater viscosity of the blood in inflamed districts as producing a slowing of the current, and Rokitansky ascribed the change to some qualitative alteration in the blood, Simon to the presence of fibrin, and Cruveilhier to the coagulation of the blood in the capillaries.

But after it had been shown that an increased velocity of the current precedes the stasis in inflammation, and Paget had observed that blood did not coagulate in occluded capillaries for a space of three days, nor, even when let out, more rapidly than other blood—a new school appeared headed by Cohnheim, who held an impairment of the walls of the vessels to be the primary cause of inflammation, in consequence of which fluid media and corpuscles could extravasate into the surrounding tissues. But this theory does not satisfactorily account for the fact that the initial acceleration of the current is diminished in the second stage and slowing obtains.

Other doctrines relating to affections of the nervous and muscular system of the vessels were advanced by Brücke, Eisenmann and Henle; the two former maintaining that spasm of the vessels, brought about either by a reflex or direct action of a stimulus, rendered the parts anæmic and caused, finally, a slowing or stoppage of the current; but the reaction after such a contraction would induce a subsequent paralysis and the current would have to become accelerated in the second stage, contrary to the actual facts.

Henle, who holds paralysis of the vessels to be the initial step towards inflammation, would have us believe that distension of a vessel causes slowing of the current, whereas this is not the case with capillaries, as Poiseuille and Jacobson have shown.

More recently theories ascribing inflammatory action to changes in the surrounding tissues, as formerly advocated by Broussai, have found followers. Virchow, indeed, believes the tissues capable of being so affected by irritants as to enable them to attract certain substances out of the blood and retain them. But these theories, again, do not sufficiently account for the circulatory phenomena.

In a lecture held before the Medical Society of Leipsic, and which has very recently appeared in pamphlet form in Volkmann's Sammlung klinischer Vorträge[1] Dr. Landerer, a brilliant young surgeon and docent of that university, from whose sketch we have taken the foregoing short survey, pointed out that in spite of the diversity of all these opinions, there is one point which bears upon all the observations made with regard to the exciting causes of inflammation, that is, that all agents capable of exciting inflammation are also capable, in a greater or less degree, of damaging or destroying the organism.

Heat, cold, traumatic injuries, chemical action, micro-organisms, stoppage of circulation—all these causes exciting inflammation, are capable, if applied with greater energy, of destroying the parts. Unbiased observation shows that, generally speaking, the tissues are first attacked by these agents, and it is only when they are introduced into the parts affected by way of the circulation, that the blood vessels and capillaries are, naturally, primarily affected. In such cases, however, the action is slower. Nor does it materially alter the course of events whether these agents are applied to paralyzed or enervated parts, showing how untenable those doctrines are which seek an explanation of inflammation in nervous affections, which are also rendered improbable since the ophthalmia occurring after section of the fifth nerve has recently been proved beyond all doubt to be simply traumatic in origin, and sympathetic ophthalmia to be due to direct invasion of micro-organisms into the healthy eye from the diseased eye by way of the sheath of the optic nerve. What elements of the tissues are first attacked by excitants of inflammation is a further point of interest. Experiments of the last few years have shown that if the blood supply to a certain organ be shut off, all elements of tissue do not perish equally rapidly; in kidneys thus rendered anæmic it is the epithelial structures, in the spinal marrow the grey matter, which is first destroyed.

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[1] No 259. "Zur Lehre von der Entzündung." Innere Medicin. No. 89. Sept. 28. 1885.

The fact that inflammation is connected with injuries to the tissues is not altogether new. The writings of Klebs, Weigert and others, and, in fact, modern bacteriology as a whole, point to this idea. But still there remains a hiatus to be filled up: to satisfactorily connect the circulatory phenomena of inflammation with this destruction of tissue. And here it is that the author advances his theory to bridge over this gap—a theory which, founded simply on the maxims of physical science, completely, in his opinion, covers all the points wherein other ones, heretofore advanced, fail, He arrives at his conclusions by the following process of reasoning:

Heretofore attention has only been paid to the fluid coursing in the capillary vessels and to the structural details of the walls of these vessels, without, however, considering if these capillaries as a whole were of themselves sufficiently firmly constructed to sustain the intra-vascular pressure alone. Now, we can more or less accurately measure, by certain experiments first performed by von Kries, the pressure inside of the capillaries, and we find that nowhere is this pressure less than 20 millimetres of mercury, or about 250 mm. of water, but frequently twice or three times that amount. But the fine endothelial structure of the capillaries alone is not capable of sustaining so great a pressure. We know that the subcutaneous und perivascular tissue-spaces also show a certain manometrical pressure, corresponding to and varying with the intravascular blood-pressure, and especially we are able to accurately measure the pressure existing in the cranial cavity exercised by the liquor cerebro-spinalis; this pressure amounts, on an average, to about 200 mm. of water. If we now average the intra-capillary pressure at 300 mm. of water, and compare it with the extra-vascular pressure of 100 mm. of water, we see that there remains the amount of 100 mm. of water to be borne by the walls of the capillaries themselves, not only in the surface of the brain, but, *mutatis mutandis*, in all parts of the body, under normal conditions. For not only do the fluids penetrating and surrounding the tissues prove capable of exerting a certain pressure or tension upon measuring instruments, but the tissues themselves all exert a tension by virtue of their natural elasticity. The author has studied this subject by means of numerous experiments, and although the figures are often small, they are not to be overlooked.

The walls of the capillaries, then, themselves unable to withstand the whole amount of the intra-vascular pressure, transfer a part of it to the surrounding intercellular fluid media and enveloping tissues, which latter are able to support the pressure by virtue of their elastic properties. For all living tissue is possessed with a very complete though not a very great elasticity; that is to say, the molecules return *exactly* to their place after having been moved apart by an application of moderate force, but they cannot return at all when a great amount of force is applied. In fact these elastic envelopes of the capillaries play the same part in turning the intermittent pump-action of the heart into the continuous movement of the capillary blood-stream, as the elastic tunic of the great arteries does with their contents.

The tissues, then, being elastic, press upon the walls of the capillaries with a pressure of about two-thirds of the intra-vascular pressure of these same capillaries—quite a considerable force.

If, now, these tissues, endowed with such elastic properties, become in any way impaired, it necessarily follows that, their function being thus damaged, changes must naturally and inevitably take place in the circulation. And as we have seen, that inflammation does impair the tissues themselves, this theory quite naturally explains how inflammation can affect the circulation, and thus bridges over the gap found in other systems.

It now only remains to show in what manner energies exciting inflammatory action can influence the elasticity of the tissues. They do so, by impairing their elasticity: the tissues are rendered inelastic, they are easily stretched and put out of shape: they are relaxed.

Thus we find the walls of inflammatory abscesses, which we have opened, shrivel up in folds; a sign that their elasticity is impaired Special measurements of portions of exsected tissue proved their elasticity diminished after they had been treated in a manner to excite inflammation by heat, chemical or bacteriological action; the author, indeed, found that inflamed tissue was comparatively very highly susceptible to tension by minute forces.

As soon as the elasticity of the tissues is diminished, the capillaries become dilated through the intra-vascular pressure, the current is accelerated, and what is called inflammatory hyperæmia obtains. At the same time the formation of lymph is increased, because the pressure within the capillaries is now greater than without. Relaxation of the tissues, however, also causes a slowing of the blood-current, as the elastic energy which before helped to impel the fluid through the capillaries, assisting the pumping action of the heart, is now missing. In this way the secondary slowing of the stream in inflamed districts is explained; especially when we consider that the amount of blood to be moved forward in the dilated capillaries is greatly increased—to as much as sixteen times the normal quantity—by the dilatation. These conditions also throw light on the efficacy of applying leeches to parts undergoing inflammation.

The present theory does not necessarily conflict with the views of Cohnheim's followers, who make chemical action affecting the vessels and tissues the prevalent means of inflammation, but it simply gives more prominence than has been hitherto done to the purely physical conduct of the parts, without touching on chemical alterations.

Œdema, which has been shown to arise from an impaired firmness and elasticity of the tissues,[1] differs from inflammation only in so far as

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[1] In a treatise published by the same author, entitled: "Ueber die Gewebsspannüng," (Leipzig, F. C. W. Vogel, 1884) which contains many points bearing upon the present theory, the following mention is made of experiments in proof of this statement:

If we make subcutaneous injections with warm wax or celloidine in healthy subjects, and subsequently, when the substance has hardened, make sections through the tumor thus obtained, we can observe that the spaces between the elements of the tisthe filaments of tissue (as can be shown under the microscope) are simply hyperextended or relaxed, whereas in inflammation the tissues are much more radically affected in their structure.

Arterial or congestive hyperæmia differs from simple inflammatory hyperæmia in that it proceeds from a paralysis of the smaller afferent vessels without the elasticity of the tissues being, at first, impaired. The energy of the current being thus increased, the current becomes accelerated while the lymph formation is not materially increased—both of which phenomena are in exact opposition to those in inflammatory hyperæmia, where the elasticity of the tissues is diminished, but the energy of the blood-current remains unaltered.

Such hyperæmiæ of a purely arterial nature are, however, of rare occurrence in man, but they may be observed after section of the nerves, in neuralgic affections, and in other nervous phenomena, as in blushing.

On the other hand, the author would have hyperæmia occurring after an application of warm water (of 40° C.) considered as identical in origin with the inflammation ensuing after more intense actions of heat.

E. H. Weber has shown that muscles in action are more easily extended than those in a state of rest; the author therefore concludes that hyperæmia in exerted muscles is, in great part, due to the diminished resistance of the tissues to the blood-supply; and furthermore, that, as organs are impaired in their constitution and damaged by the very exercise of their function, the hyperæmia induced by such damage, affecting their elastic properties as well, does not qualitatively differ from what is generally called reparative inflammation.

Inflammation, in fact, far from being an enemy to the organization of the body, is essential to its health; it is a weapon with which the body combats destructive influences in healthy parts. The heightened afflux of blood to inflamed parts and their copious permeation, we might say irrigation, with lymph-fluid, serve to cleanse the tissues of

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sues are all distended and filled with the substance injected—just as they are pathologically distended by air in subcutaneous emphysema or with pus in phlegmone. And under the microscope these sections present *regular rhomboid figures*. If, however, we in the same manner inject parts affected with *œdema* and examine the preparation in the same way, the interstices will not, as before, present rhomboid figures, but they will appear *perfectly round* in every section, thus representing balls.

deleterious substances and to convey elements of reconstruction to where they may be needed.

Turning from theory to practice, the author believes it imprudent to combat inflammation, which is, in reality, a reparatory process; or, at least, it is futile. For traumatic inflammation he favors warmth and moist applications. Ice is only of value in producing local anæsthesia in fractures, etc., but retards the healing process. In conclusion, the author adds several interesting and ingenious hypotheses in support of his theories.

As to the emigration of white blood-corpuscles observed in inflammation, the author considers this phenomenon capable of being brought into connection with his theory of regarding inflammation as a reparatory process, and appears inclined to believe, in keeping with the most recent observations, that the leucocytes emigrate of their own accord and battle with infective agents.

As to the differentiation of various inflammatory processes and their classification as serous, purulent, croupous, cheesy, and other species of inflammation, he inclines to the idea, now fast gaining ground, that each separate type of inflammation is due to distinct exciting causes, each one calling forth its own peculiar form of specific inflammation.

The fact that inflammation occasioned by bacteria should produce suppuration instead of some other form of inflammation, the author believes to be analogous to the liquefaction of the gelatine in cultures; perhaps the production of peptone can account for the failing coagulation. Thus tubercle-bacilli do not liquefy the media of cultivation, and in the body they form cheesy abscesses.

That leucocytes should be so sparingly found in traumatic inflammation the author attributes to the high tension existing in the tissues, due to the numerous smaller hæmorrhages. W. VAN ARSDALE.